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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.
09/531,969	03/21/00	GELIEBTER J	96/007596

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EXAMINER	
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ART UNIT	**PAPER NUMBER**
1632	4

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Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

Office Action Summary	Application No.	Applicant(s)	
	09/531,969	GELIEBTER ET AL.	
	Examiner	Art Unit	
	Peter Paras	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136 (a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☐ Responsive to communication(s) filed on _____ .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-36* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-36* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claims _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are objected to by the Examiner.

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).

Attachment(s)

15) ☒ Notice of References Cited (PTO-892)
16) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
17) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____ .
18) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
19) ☐ Notice of Informal Patent Application (PTO-152)
20) ☐ Other: .

DETAILED ACTION

Claims 1-36 are pending.

Claim Rejections - 35 USC § 112, 1st paragraph

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> **The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.**

Claims 1-8, 10-18, and 20 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The claims are directed to a method of regulating smooth muscle tone in a subject, comprising introduction and expression of a DNA sequence encoding a protein involved in the regulation of smooth muscle tone in a sufficient number of smooth muscle cells. The claims are also directed to a vector comprising a nucleotide sequence encoding a protein involved in the regulation of smooth muscle tone and a smooth muscle cell comprising the same nucleotide sequence.

Vas-Cath Inc. v. Mahurkar, 19USPQ2d 1111 (Fed. Cir. 1991), clearly states that "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession *of the invention*. The invention is, for purposes of the 'written description' inquiry, *whatever is now claimed*." Vas-Cath Inc. v. Mahurkar, 19USPQ2d at 1117. The specification does not "clearly allow persons of

ordinary skill in the art to recognize that [he or she] invented what is claimed."Vas-Cath Inc. v. Mahurkar, 19USPQ2d at 1116.

Applicants invention is drawn to methods of regulating smooth muscle tone by introduction of a nucleic acid sequence into smooth muscle cells such that the nucleic acid sequence is expressed and smooth muscle tone is regulated. The broadest claims are directed to methods of regulating smooth muscle tone by introduction of any nucleic acid sequence into any smooth muscle cells. While the specification has described methods of regulating penile and bladder smooth muscle tone by direct injection of nucleic acid sequences encoding Maxi-K and a method of regulating penile smooth muscle tone by direct injection of a nucleic acid sequence encoding Kir6.2, the specification has not described regulation of smooth muscle tone by introduction of any other nucleic acid sequences. The specification fails to describe other nucleic acid sequences which when introduced and expressed into smooth muscle cells result in regulation of smooth muscle tone. Additionally, the state of the art was such that *in vivo* expression of nucleic acid molecules, other than Maxi-K and Kir6.2 as described above, which resulted regulation of smooth muscle tone were not known. The claimed invention as a whole is not adequately described if the claims require essential or critical elements which are not adequately described in the specification and which are not conventional in the art **as of Applicants effective filing date**. Possession may be shown by actual reduction to practice, clear depiction of the invention in a detailed drawing, or by describing the invention with sufficient relevant identifying characteristics (as it relates to the claimed invention as a whole) such that a person skilled in the art

would recognize that the inventor had possession of the claimed invention. Pfaff v. Wells Electronics, Inc., 48 USPQ2d 1641, 1646 (1998). In the instant case, methods of regulating smooth muscle tone using nucleic acid sequences other than Maxi-K or Kir6.2 that regulate smooth muscle tone lack a written description. The specification fails to describe what other genes fall into this genus and it was unknown as of Applicants' effective filing date that any of these genes would have the properties of regulating smooth muscle tone. The skilled artisan cannot envision the method steps necessary to practice the claimed methods, and therefore conception is not achieved until reduction to practice has occurred, regardless of the complexity or simplicity of the method. Adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method of isolating it. See *Fiers v. Revel*, 25 USPQ2d 1601, 1606 (Fed. Cir. 1993) and *Amgen Inc. v. Chugai Pharmaceutical Co. Ltd.*, 18 USPQ2d 1016 (Fed. Cir. 1991).

One cannot describe what one has not conceived. See *Fiddes v. Baird*, 30 USPQ2d 1481, 1483. In *Fiddes*, claims directed to mammalian FGF's were found to be unpatentable due to lack of written description for that broad class. The specification provided only the bovine sequence.

Applicant is reminded that *Vas-Cath* makes clear that the written description provision of 35 U.S.C. §112 is severable from its enablement provision (see page 1115).

Claims 1-20 and 28-36 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of regulating bladder smooth muscle tone or treatment of erectile dysfunction caused by heightened contractility of smooth muscle by direct injection of a nucleotide sequence encoding Maxi-K wherein expression of Maxi-K results in less heightened contractility, and a method of treatment of erectile dysfunction caused by heightened contractility of smooth muscle by direct injection of nucleotide sequence encoding the Kir 6.2 K_{ATP} subunit wherein expression of Kir 6.2 results in less heightened contractility, and isolated smooth muscle cells transformed *in vitro* with the same nucleic acid sequences that may regulate smooth muscle tone, does not reasonably provide enablement for a method of regulating smooth muscle tone *in vivo* by introduction of other nucleotide sequences that encode proteins which may be involved in the regulation of smooth muscle tone, or other smooth muscle cells *in vivo* (not-isolated) comprising an exogenous nucleotide sequence that may regulate smooth muscle tone. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The claims are directed to regulation of smooth muscle tone by introduction of a nucleotide sequence encoding a protein that may be involved in the regulation of smooth muscle tone. The claims are also directed to a smooth muscle cell (not isolated) comprising an exogenous nucleotide sequence encoding a protein that may be involved in the regulation of smooth muscle tone.

The specification discusses that the invention features methods of gene therapy for regulating smooth muscle tone through delivery of a DNA sequence encoding a protein involved in the regulation of smooth muscle tone into a smooth muscle cell (page 8 lines 13-17). The specification discusses that the invention features methods of gene therapy used to treat erectile dysfunction and separate methods of gene therapy to alleviate bladder dysfunction (see pages 4-9). The specification provides extensive teachings pertaining to the treatment of erectile and bladder dysfunction caused by heightened contractility of smooth muscle by direct injection of a nucleotide sequence encoding MaxiK into penile and bladder smooth muscle cells in respective art-recognized models. The specification has also provided extensive teachings relating to the treatment of erectile dysfunction caused by heightened contractility of smooth muscle cells by direct injection of a nucleotide sequence encoding the Kir6.2 K_{ATP} subunit. See pages 51-74 of the specification. The specification however fails to provide relevant teachings or guidance, or working examples that correlates the delivery of the nucleotide sequences that encode the other recited proteins with regulation of smooth muscle tone.

As a first issue, with regard to claim breadth, while the specification has provided working examples that demonstrate treatment of bladder dysfunction and erectile dysfunction by direct injection of a nucleotide sequence encoding MaxiK and treatment of erectile dysfunction by direct injection of a nucleotide sequence encoding the Kir6.2 K_{ATP} subunit as noted above; it fails to provide sufficient guidance and/or evidence that would correlate to the introduction into smooth muscle of nucleotide sequences

encoding the other recited proteins. The specification has not correlated the results obtained with MaxiK and Kir6.2 with the other recited proteins regarding regulation of smooth muscle tone. It is unpredictable if any nucleotide sequence encoding the any of the other recited proteins, when introduced by any route of administration into dysfunctional smooth muscle cells can regulate smooth muscle tone, wherein the regulation results in heightened or less heightened contractility of smooth muscle cells. [Interestingly, the recited proteins come from different protein families, for example MaxiK and Kir6.2 are members of a potassium channel protein family, while the other recited proteins come from other protein families like calcium channels, kinases, endothelins. The relationship between the different classes of recited proteins is unclear regarding regulation of smooth muscle tone. Furthermore, the relationship between the recited dysfunctions such as asthma, hyperplasia of the prostate gland coronary artery disease genitourinary dysfunction, irritable bowel syndrome, migraine headaches, premature labor, Raynaud's syndrome, and thromboangitis obliterans and the recited proteins is unclear. The specification has not established a correlation between the various recited proteins and the recited dysfunctions so it is unclear how the recited proteins could treat the recited dysfunctions.] It is well known that the status of the gene therapy art, in particular, at the time of the effective filing date of the claimed invention, was undeveloped and unpredictable in terms of achieving *in vivo* therapeutic expression levels of a gene of interest. See Eck & Wilson, who (The Pharmacological Basis of Therapeutics, 1995) report that numerous factors complicate *in vivo* gene therapy with respect to predictably achieving levels and duration of gene expression

which have not been shown to be overcome by routine experimentation. These include, the fate of the DNA vector itself (volume distribution, rate of clearance into the tissues, etc.), the *in vivo* consequences of altered gene expression and protein function, the fraction of vector taken up by the target cell population, the trafficking of the genetic material within cellular organelles, the rate of degradation of the DNA, the level of mRNA produced, the stability of the mRNA produced, the amount and stability of the protein produced, and the protein's compartmentalization within the cell, or its secretory fate, once produced. See Eck and Wilson, 1995, page 82, column 1, first paragraph. These factors differ dramatically based on the protein being produced, and the disease and/or host being treated. It is further noted that Eck and Wilson supports the importance of tailoring a gene therapy vector and method to specific diseases and/or disorders and not to all diseases and disorders. See page 82, column 1, first paragraph. For example, Eck & Wilson et al. review the state of the art for gene therapy for inherited disorders and discloses that "[t]he level of protein function necessary to achieve complementation of the defect varies widely among genetic diseases." See page 78, column 2, 2nd paragraph. As such, in light of the state of the art for gene therapy, Applicants fail to provide guidance for the above parameters for *in vivo* gene expression nor do they provide a clear correlation to carrying out methods utilizing the contemplated genes, other than Kir6.2 or MaxiK, of the invention to achieve gene therapy with regard to any particular therapeutic effect with regard to regulation of smooth muscle tone.

To this regard, MPEP section 2164 sets forth that the issue of "correlation" is also dependent on the state of the art at the time of the invention. MPEP, section 2164 goes on to discuss that if one skilled in the art cannot readily anticipate the effect of a change within the subject matter to which that claimed invention broadly pertains, then there is lack of predictability in the art. Thus, what is known in the art provides evidence as to the question of predictability.

The claims also encompass various means of gene delivery and subsequent cell targeting. While progress has been made in recent years for gene transfer *in vivo*, vector targeting to desired tissues *in vivo* continues to be unpredictable and inefficient as supported by numerous teachings available in the art. For example, Miller (1995, FASEB J., Vol. 9, pages 190-199) review the types of vectors available for *in vivo* gene therapy, and conclude that "for the long-term success as well as the widespread applicability of human gene therapy, there will have to be advances...targeting strategies outlined in this review, which are currently only at the experimental level, will have to be translated into components of safe and highly efficient delivery systems" (page 198, column 1). Deonarain (1998, Expert Opin. Ther. Pat., Vol. 8, pages 53-69) indicate that one of the biggest problems hampering successful gene therapy is the "ability to target a gene to a significant population of cells and express it at adequate levels for a long enough period of time" (page 53, first paragraph). Deonarain reviews new techniques under experimentation in the art which show promise but states that such techniques are even less efficient than viral gene delivery (see page 65, first paragraph under Conclusion section). Verma (Sept. 1997, Nature, Vol. 389, pages 239-

242) reviews vectors known in the art for use in gene therapy and discusses problems associated with each type of vector. The teachings of Verma indicate a resolution to vector targeting has not been achieved in the art (see entire article). Verma also teaches appropriate regulatory elements may improve expression, but it is unpredictable what tissues such regulatory elements target (page 240, sentence bridging columns 2 and 3). Crystal (1995, Science, Vol. 270, page 404-410) also reviews various vectors known in the art and indicates that "among the design hurdles for all vectors are the need to increase the efficiency of gene transfer, to increase target specificity and to enable the transferred gene to be regulated" (page 409).

As such, the specification is enabling only for methods of regulating penile or bladder smooth muscle tone by direct injection of a nucleic acid sequence encoding Maxi-K or for a method of regulating penile smooth muscle tone by direct injection of a nucleic acid seqeunce encoding Kir6.2.

With regard to claims 28-36 which are drawn to smooth muscle cells comprising an exogenous DNA sequence encoding a protein involved in the regulation of smooth muscle tone, the specification does not enable smooth muscle cells *in vivo* comprising exogenous DNA sequences encoding a protein involved in the regulation of smooth muscle tone for the reasons provided above with regard to routes of administration, expression levels resulting in regulation of smooth muscle tone, vectors, and targeting. This rejection may be overcome if the claims were amended to be directed to an isolated cell.

Accordingly, in view of the quantity of experimentation necessary to determine the parameters listed above, the lack of direction or guidance provided by the specification to carry out gene therapy for regulation of smooth muscle tone as broadly claimed using the recited genes, the absence of working examples for the demonstration or correlation to achieving levels of therapeutic expression of a heterologous gene *in vivo*, the breadth of the claims, and the unpredictable and undeveloped state of the art with respect to the gene therapy art and cell targeting, it would have required undue experimentation for one skilled in the art to carry out the claimed methods without a reasonable expectation of success.

Claim Rejections - 35 USC § 112, 2nd paragraph

The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 7-19, 21-27, and 30-34 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 12 and 14 are indefinite as written. The claims are directed to various proteins that modulate contraction of smooth muscle but also recite diacylglycerol, which is not a protein. The metes and bounds of the claims cannot be determined as the claim is directed to proteins that modulate contraction of smooth muscle but recite proteins and non-proteins.

In claim 21, the limitation "capable of" renders the claim indefinite because the metes and bounds of the claim cannot be determined. Claim 21 is directed to a recombinant vector comprising a portion of a viral genome. It is unclear what elements are part of the portion of the viral genome which render it necessary to perform the required function. Claims 22-27 depend from claim 21.

The term "modulates" in claims 7, 10-11, 13, 15-19, 22, 25, 30, 33, and 34 is a relative term which renders the claim indefinite. The term "modulates" is not defined by the claim, the specification does not provide a standard for ascertaining the requisite degree, and one of ordinary skill in the art would not be reasonably apprised of the scope of the invention. The meaning of modulate is unclear from the specification. The claims as written do not define "modulate". It is unclear how smooth muscle is modulated by expression of the recited proteins. For example, it is unclear how expression of the proteins can "modulate" relaxation of smooth muscle. Claims 8-9, 12, 14, 23-24, 26, and 31-32 depend from the rejected claims.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

(e) the invention was described in a patent granted on an application for patent by another filed in the United States before the invention thereof by the applicant for patent, or on an international application by another who has fulfilled the requirements of paragraphs (1), (2), and (4) of section 371(c) of this title before the invention thereof by the applicant for patent.

Claims 21-25, 27-33, and 35-36 are rejected under 35 U.S.C. 102(e) as being anticipated by Kaczorowski (US 5,776,734).

The claims are directed to a recombinant vector comprising a viral regulatory sequence and a nucleotide sequence encoding a protein involved in regulating smooth muscle tone. The claims are also directed to a smooth muscle cell comprising and expressing an exogenous nucleotide sequence that encodes a protein involved in regulating smooth muscle tone.

Kaczorowski et al teach the isolation and cloning of the nucleotide sequence that encodes the β-subunit of the Maxi-K potassium channel from bovine tracheal smooth muscle cells. See Example 1, columns 15-18. Kaczorowski teaches that Maxi-K regulates smooth muscle tone. Kaczorowski teaches smooth muscle cells transfected with a viral vector encoding Maxi-K. See column 9 lines 29-45. The viral vectors may be retroviral, adenoviral, adeno-associated virus, or herpes. See column 12. Kaczorowski et al also teach that an expression vector comprising a nucleotide sequence that encodes Maxi-K may be introduced into host cells by transformation, transfection, lipofection, protoplast fusion, or electoporation (see column 9). Thus, the teachings of Kaczorowski meet all of the instant claim limitations.

Claims 21-23, 27-31, and 35-36 are rejected under 35 U.S.C. 102(e) as being anticipated by Kalecko et al (US 6,156,497).

Kalecko et al teaches adenoviral vectors comprising internal terminal repeats (ITRs) that direct expression of a heterologous gene. See columns 7, 13, and examples 1-4. Kalecko teaches that the heterologous gene can be nitric oxide synthase or a vascular muscle calcium channel. Kalecko teaches that vascular smooth muscle cells are transfected. See column 10. Thus, the teachings of Kalecko meet all of the instant claim limitations.

Claims 21, 25-27, 28, and 33-34 are rejected under 35 U.S.C. 102(b) as being anticipated by Yoshitaka et al (US 5,219,748, June 15, 1993).

The claims are directed to a vector comprising part of a virus genome that can direct expression of a heterologous gene. The claims are also directed to the same vector wherein the heterologous gene is protein kinase C.

Yoshitaka et al teach the nucleic acid sequence encoding human and rat protein kinase C. See examples 1-4 in columns 10-17. Yoshitaka also teach that the nucleotide sequence encoding protein kinase C may be cloned into a retroviral vector. See column 6. Yoshitaka teach transfected smooth muscle cells comprising a nucleic acid sequence encoding protein kinase C. See column 7. Thus, the teachings of Yoshitaka meet all of the instant claim limitations.

Doubl Pat nting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. See *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970);and, *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.130(b).

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 1-15, 17-18, and 20 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-9 of U.S. Patent No. 6,150,338. Although the conflicting claims are not identical, they are not patentably distinct from each other because both sets of claims are directed to methods of regulating smooth muscle tone, wherein a nucleotide sequence encoding a protein that regulates smooth muscle tone is introduced into smooth muscle by direct injection, particularly when the nucleic acid sequence encodes Maxi-K and is introduced into penile smooth muscle to treat erectile dysfunction caused by heightened contractility, and wherein expression of Maxi-K results in less heightened contractility.

Claims 1-15 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-2 of U.S. Patent No. 6,239,177. Although the conflicting claims are not identical, they are not patentably distinct from each other because both sets of claims are directed to methods of

regulating smooth muscle tone, wherein a nucleotide sequence encoding a protein that regulates smooth muscle tone is introduced into smooth muscle by direct injection, particularly when the nucleic acid sequence encodes Maxi-K and is introduced into bladder smooth muscle to treat bladder dysfunction caused by heightened contractility, and wherein expression of Maxi-K results in less heightened contractility.

Conclusion

No claims are allowed. Claims 16 and 19 appear to be free of the prior art of record because the prior art of record does not teach or suggest methods of regulating smooth muscle tone comprising introducing a nucleic acid sequence encoding protein kinase C or connexin 43, wherein the nucleic acid sequences are expressed and smooth muscle tone is regulated.

Any inquiry concerning this communication or earlier communications from the examiner(s) should be directed to Peter Paras, Jr., whose telephone number is 703-308-8340. The examiner can normally be reached Monday-Friday from 8:30 to 4:30 (Eastern time).

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Karen Hauda, can be reached at 703-305-6608. Papers related to this application may be submitted by facsimile transmission. Papers should be faxed via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center numbers are (703)308-4242 and (703)305-3014.

Inquiries of a general nature or relating to the status of the application should be directed to Kay Pinkney whose telephone number is (703) 305-3553.

Peter Paras, Jr.

Art Unit 1632

JILL D. MARTIN
PRIMARY EXAMINER